

THE OILING OF AMERICA

Modern-day diets high in hydrogenated vegetable oils instead of traditional animal fats are implicated in causing a significant increase in heart disease and cancer.

Part 1 of 2

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In 1954, a young researcher from Russia, named David Kritchevsky, published a paper describing the effects of feeding cholesterol to rabbits.¹ Cholesterol added to vegetarian rabbit chow caused the formation of atheromas—plaques that block arteries and contribute to heart disease. Cholesterol is a heavyweight molecule—an alcohol or a sterol—found only in animal foods such as meat, cheese, eggs and butter.

In the same year, according to the American Oil Chemists Society, Kritchevsky published a paper describing the beneficial effects of polyunsaturated fatty acids for lowering cholesterol levels.² (Polyunsaturated fatty acids are the kind of fats found in large amounts in highly liquid vegetable oils made from corn, soybeans, safflower seeds and sunflower seeds. Mono-unsaturated fatty acids are found in large amounts in olive oil, palm oil and lard; saturated fatty acids are found in large amounts in fats and oils that are solid at room temperature, e.g., butter, tallow and coconut oil.)

Scientists of the period were grappling with a new threat to public health: a steep rise in heart disease. While turn-of-the-century mortality statistics are unreliable, they consistently indicate that heart disease caused no more than 10 per cent of all deaths—considerably less than infectious diseases such as pneumonia and tuberculosis. By 1950, coronary heart disease (CHD) was the leading source of mortality in the United States, causing more than 30 per cent of all deaths. The greatest increase came under the rubric of myocardial infarction (MI)—a massive blood clot leading to obstruction of a coronary artery and consequent death to the heart muscle. MI was almost non-existent in 1910 and caused no more than 3,000 deaths per year in 1930. By 1960, there were at least 500,000 MI deaths per year in the US. What lifestyle changes had caused this increase?

One change was a decrease in infectious disease following the decline of the horse as a means of transport, the installation of more sanitary water supplies and the advent of better housing, all of which allowed more people to reach adulthood and the heart attack age. The other was a dietary change.

Since the early part of the century when the US Department of Agriculture (USDA) had begun to keep track of food 'disappearance' data (the amount of various foods going into the food supply), a number of researchers had noticed a change in the kind of fats Americans were eating. Butter consumption was declining, while the use of vegetable oils, especially oils that had been hardened to resemble butter by a process called 'hydrogenation', was increasing dramatically. By 1950, butter consumption had dropped from 18 pounds per person per year to just over 10 pounds. Margarine filled in the gap, rising from about 2 pounds per person at the turn of the century to about 8 pounds. Consumption of vegetable shortening—used in crackers and baked goods—remained relatively steady at about 12 pounds per person per year, but vegetable oil consumption had more than tripled from just under 3 pounds per person per year to more than 10 pounds.³

The statistics pointed to one obvious conclusion: Americans should eat the traditional foods—including meat, eggs, butter and cheese—that nourished their ancestors, and avoid the newfangled, vegetable-oil-based foods that were flooding the grocers' shelves.

The Kritchevsky articles attracted immediate attention because they lent support to another theory—one that militated against the consumption of meat and dairy products. This was the lipid hypothesis: namely, that saturated fat and cholesterol from animal sources raise cholesterol levels in the blood, leading to deposition of cholesterol and fatty material as pathogenic plaques in the arteries.

Kritchevsky's rabbit trials were actually a repeat of studies carried out four decades earlier in St Petersburg, in which rabbits fed saturated fats and cholesterol developed fatty

deposits in their skin and other tissues—and in their arteries. By showing that polyunsaturated oils from vegetable sources lowered serum cholesterol at least temporarily in humans, Kritchevsky appeared to show that the findings from the animal trials were relevant to the CHD problem, that the lipid hypothesis was a valid explanation for the new epidemic, and that, by reducing animal products in their diets, Americans could avoid heart disease.

In the years that followed, a number of population studies demonstrated that the animal model—especially one derived from vegetarian animals—was not a valid approach for the problem of heart disease in human omnivores.

A 1955 report on artery plaques in soldiers killed during the Korean War showed little difference in the number and severity of plaques between American soldiers and those of Japanese natives—75 per cent versus 65 per cent—even though the Japanese diet at the time was lower in animal products and fat.⁴ A 1957 study of the largely vegetarian Bantu found that they had as much atheroma—occlusions or plaque build-up in the arteries—as other races from South Africa who ate more meat.⁵ A 1959 report noted that Jamaican Blacks showed a degree of atherosclerosis comparable to that found in the United States, although they suffered from lower rates of heart disease.⁶ A 1960 report noted that the severity of atherosclerotic lesions in Japan approached that of the United States.⁷ The 1968 International Atherosclerosis Project, in which over 22,000 corpses in 14 nations were cut open and examined for plaques in the arteries, showed the same degree of atheroma in all parts of the world—in populations that suffered from a great deal of heart disease, and in populations that had very little or none at all.⁸

All of these studies pointed to the fact that the thickening of the arterial walls is a natural, unavoidable process. The lipid hypothesis did not hold up to these population studies, nor did it explain the tendency toward fatal clots that caused myocardial infarction.

In 1956, an American Heart Association (AHA) fund-raiser was aired on all three major networks. The Master of Ceremonies interviewed, among others, Irving Page and Jeremiah Stamler of the AHA and researcher Ancel Keys. Panellists presented the lipid hypothesis as the cause of the heart disease epidemic and launched the Prudent Diet, one in which corn oil, margarine, chicken and cold cereal replaced butter, lard, beef and eggs.

The television campaign was not an unqualified success because one of the panellists, Dr Dudley White, disputed his colleagues at the AHA. Dr White noted that heart disease in the form of myocardial infarction was non-existent in 1900 when egg consumption was three times what it was in 1956 and when corn oil was unavailable. When pressed to support the Prudent Diet, Dr White replied: "See here, I began my practice as a cardiologist in 1921 and I never saw an MI patient until 1928. Back in the MI-free days before 1920, the fats were butter and lard, and I think that we would all benefit from the kind of diet that we had at a time when no one had ever heard the word *corn* oil."

But the lipid hypothesis had already gained enough momentum to keep it rolling, in spite of Dr White's nationally televised plea for common sense in matters of diet and in spite of the contradictory studies that were showing up in the scientific literature.

In 1957, Dr Norman Jolliffe, Director of the Nutrition Bureau of the New York Health Department, initiated the Anti-Coronary Club in which selected businessmen, ranging in age from 40 to 59 years, were placed on the Prudent Diet. Club members used corn oil and margarine instead of butter; cold breakfast cereals instead of eggs and chicken; and fish instead of beef. Anti-Coronary Club members were to be compared with a 'matched' group of the same age who ate eggs for breakfast and had meat three times a day. Dr Jolliffe, an overweight diabetic confined to a wheelchair, was confident that the Prudent Diet would save lives, including his own.

In the same year, the food industry initiated advertising campaigns that touted the health benefits of their products: "Low in fat" or "Made with vegetable oils". A typical ad read, "Wheaties may help you live longer". Wesson recommended its cooking oil "for your heart's sake". An ad in the *Journal of the American Medical Association (JAMA)* described Wesson oil as a "cholesterol depressant". Mazola advertisements assured the public that "science finds corn oil important to your health". Medical journal ads recommended Fleishmann's unsalted margarine for patients with high blood pressure.

In his syndicated column, Dr Frederick Stare, head of Harvard University's Nutrition Department, encouraged the consumption of corn oil—up to one cup a day. In a promotional piece specifically for Procter & Gamble's Puritan oil, he cited two experiments and one clinical trial as showing that high blood cholesterol is

associated with CHD. However, both experiments had nothing to do with CHD, and the clinical trial did not find that reducing blood cholesterol had any effect on CHD events. Later, Dr William Castelli, director of the Framingham Study, was one of several specialists to endorse Puritan. Dr Antonio Gotto, Jr, former AHA president, sent practising physicians a letter promoting Puritan oil—printed on Baylor College of Medicine, The De Bakey Heart Center letterhead.⁹

The irony of Gotto's letter is that De Bakey, the famous heart surgeon, co-authored a 1964 study involving 1,700 patients, which also showed no definite correlation between serum cholesterol levels and the nature and extent of coronary artery disease.¹⁰ In other words, those with low cholesterol levels were just as likely to have blocked arteries as those with high cholesterol levels.

But while studies like DeBakey's mouldered in the basements of university libraries, the vegetable oil campaign took on increased bravado and audacity.

The American Medical Association (AMA) at first opposed the commercialisation of the lipid hypothesis and warned that "the anti-fat, anti-cholesterol fad is not just foolish and futile...it also carries some risk".

The American Heart Association, however, was committed. In 1961, the AHA published its first dietary guidelines aimed at the public. The authors—Irving Page, Ancel Keys, Jeremiah Stamler and Frederick Stare—called for the substitution of polyunsaturated for saturated fat, even though Keys, Stare and Page had all previously noted in published papers that the increase in CHD was paralleled by increasing consumption of vegetable oils. In fact, in a 1956 paper, Keys had suggested that the increasing use of hydrogenated vegetable oils might be the underlying cause of the CHD epidemic.¹¹

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Stamler showed up again in 1966 as an author of *Your Heart Has Nine Lives*, a little self-help book advocating the substitution of vegetable oils for butter and other so-called 'artery-clogging' saturated fats. The book was sponsored by the makers of Mazola corn oil and margarine. Stamler did not believe that lack of evidence should deter Americans from changing their eating habits. The evidence, he stated, was "compelling enough to call for altering some habits even before the final proof is nailed down...the definitive proof that middle-aged men who reduce their blood cholesterol will actually have far fewer heart attacks waits upon diet studies now in progress". His version of the Prudent Diet called for substituting low-fat milk products such as skim milk and low-fat cheeses for cream, butter and whole cheeses, reducing egg consumption and cutting the fat off red meats. Heart disease, he lectured, was a disease of rich countries, striking rich people who ate rich food, including 'hard' fats like butter.

It was in the same year, 1966, that the results of Dr Jolliffe's Anti-Coronary Club experiment were published in *JAMA*.¹² Those on the Prudent Diet of corn oil, margarine, fish, chicken and cold cereal had an average serum cholesterol of 220, compared to 250 in the meat-and-potatoes control group. However, the study authors were obliged to note that there were eight deaths from heart disease among Dr Jolliffe's Prudent Diet group, and none among those who ate meat three times a day. Dr Jolliffe was dead by this time. He succumbed in 1961 to a vascular thrombosis, although the obituaries listed the cause of death as "complications from diabetes". The compelling "proof" that Stamler and others were sure would vindicate wholesale tampering with American eating habits had not yet been "nailed down".

The problem, said the insiders promoting the lipid hypothesis, was that the numbers involved in the Anti-Coronary Club experiment were too small. Dr Irving Page urged a National Diet-Heart Study involving one million men, in which the results of the Prudent Diet could be compared on a large scale with those on a diet high in meat and fat. With great media attention, the National Heart, Lung and Blood Institute organised the stocking of food warehouses in six major cities, where men on the Prudent Diet could get tasty polyunsaturated doughnuts and other fabricated food items free of charge.

But a pilot study, involving 2,000 men, resulted in exactly the same number of deaths in both the Prudent Diet group and control group. A brief report in *Circulation* (March 1968) stated that the study was a milestone "in mass environmental experimentation" that would have "an important effect on the food industry and the attitude of the public toward its eating habits". But the million-man Diet-Heart Study was abandoned in utter silence "for reasons of cost". Its chairman, Dr Irving Page, died of a heart attack.

Most animal fats—like butter, lard and tallow—have a large proportion of saturated fatty acids. Saturated fats are straight chains of carbon and hydrogen that pack together easily so that they are relatively solid at room temperature. Oils from seeds are composed mostly of polyunsaturated fatty acids. These molecules have kinks in them at the point of the unsaturated double bond. They do not pack together easily and therefore tend to be liquid at room temperature.

Judging from both food data and turn-of-the-century cookbooks, the American diet in 1900 was a rich one, with at least 35 to 40 per cent of calories coming from fats, mostly dairy fats in the form of butter, cream, whole milk, and also eggs. Salad dressing recipes usually called for egg yolks or cream; only occasionally for olive oil. Lard or tallow served for frying. Rich dishes like head cheese and scrapple contributed additional saturated fats during an era when cancer and heart disease were rare. Butter substitutes made up only a small portion of the American diet, and these margarines were blended from coconut oil, animal tallow and lard—all rich in natural saturates.

The technology by which liquid vegetable oils could be hardened to make margarine was first discovered by a French chemist named Sabatier. He found that a nickel catalyst would cause the hydrogenation (the addition of hydrogen to unsaturated bonds to make them saturated) of ethylene gas to ethane. Subsequently, the British chemist Norman developed the first application of hydrogenation to food oils and took out a patent.

In 1909, Procter & Gamble acquired the US rights to a British patent on making liquid vegetable oils solid at room temperature. The process was used on both cotton-seed oil and lard to give

"better physical properties", to create shortenings that did not melt as easily on hot days.

The hydrogenation process transforms unsaturated oils into straight 'packable' molecules by rearranging the hydrogen atoms at the double bonds. In nature, most double bonds occur in the *cis* configuration, i.e., with both hydrogen atoms on the same side of the carbon chain at the point of the double bond. It is the *cis* isomers of fatty acids that have a bend or kink at the double bond, preventing them from packing together easily. Hydrogenation creates *trans*

double bonds by moving one hydrogen atom across to the other side of the carbon chain at the point of the double bond. In effect, the two hydrogen atoms then balance each other and the fatty acid straightens, creating a packable 'plastic' fat with a much higher melting temperature.

Although *trans* fatty acids are technically unsaturated, they are configured in such a way that the benefits of unsaturation are lost. The presence of several unpaired electrons presented by contiguous hydrogen atoms in their *cis* form allows many vital chemical reactions to occur at the site of the double bond. When one hydrogen atom is moved to the other side of the fatty acid molecule during hydrogenation, the ability of living cells to make reactions at the site is compromised or altogether lost. *Trans* fatty acids are sufficiently similar to natural fats that the body readily incorporates them into the cell membrane; once there, their altered chemical structure creates havoc with thousands of necessary chemical reactions—everything from energy provision to prostaglandin production.

After the Second World War, 'improvements' made it possible to plasticise highly unsaturated oils from corn and soybeans. New catalysts allowed processors to 'selectively hydrogenate' the kinds of fatty acids found in soy and canola oils—those with three double bonds. Called 'partial hydrogenation', this new method allowed processors to replace cotton-seed oil with more unsaturated corn and soybean oils in margarines and shortenings. This spurred a meteoric rise in soybean production from virtually

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nothing in 1900 to 70 million tons in 1970, surpassing corn production. Today, soy oil dominates the market and is used in almost 80 per cent of all hydrogenated oils.

The particular mix of fatty acids in soy oil results in shortenings containing about 40 per cent *trans* fats—an increase of about 5 per cent over cotton-seed oil and 15 per cent over corn oil. Canola oil, processed from a hybrid form of rape-seed, is particularly rich in fatty acids containing three double bonds and can contain as much as 50 per cent *trans* fats. *Trans* fats of a particularly problematic type are also formed during the process of deodorising canola oil, yet they are not indicated on labels for canola oil.

Certain forms of *trans* fatty acids occur naturally in dairy fats. *Trans* vaccenic acid makes up about four per cent of the fatty acids in butter. It is an interim product which the ruminant animal then converts to conjugated linoleic acid, a highly beneficial anti-carcinogenic component of animal fat. Humans seem to utilise the small amounts of *trans* vaccenic acid in butter fat without ill effects.

However, most of the *trans* isomers in modern hydrogenated fats are new to the human physiology. By the early 1970s, a number of researchers had expressed concern about their presence in the American diet, noting that the increasing use of hydrogenated fats had paralleled the increase in both heart disease and cancer. The unstated solution was one that could be easily presented to the public: eat natural, traditional fats; avoid newfangled foods made from vegetable oils; use butter, not margarine.

But medical research and public consciousness took a different tack—one that accelerated the decline of traditional foods like meat, eggs and butter, and fuelled continued dramatic increases in vegetable oil consumption.

Although the AHA had committed itself to the lipid hypothesis and the unproven theory that polyunsaturated oils afforded protection against heart disease, concerns about hydrogenated vegetable oils were sufficiently great to warrant the inclusion of the following statement in the organisation's 1968 Diet-Heart statement: "Partial hydrogenation of polyunsaturated fats results in the formation of *trans* forms which are less effective than *cis,cis* forms in lowering cholesterol concentrations. It should be noted that many currently available shortenings and margarines are partially hydrogenated and may contain little polyunsaturated fat of the natural *cis,cis* form."

While 150,000 copies of the statement were printed, they were never distributed. The shortening industry objected strongly, and a researcher named Fred Mattson of Procter & Gamble convinced Campbell Moses, medical director of the AHA, to remove it.¹³ The final recommendations for the public contained three major points: restrict calories; substitute polyunsaturates for saturates; reduce cholesterol in the diet.

Other organisations fell in behind the AHA in pushing vegetable oils instead of animal fats. By the early 1970s, the National Heart, Lung and Blood Institute, the AMA, the American Dietetic Association and the National Academy of Sciences had all endorsed the lipid hypothesis and the avoidance of animal fats for those Americans in the 'at risk' category.

Since Kritchevsky's early studies, many other trials had shown that serum cholesterol can be lowered by increasing ingestion of polyunsaturates. The physiological explanation for this is that when excess polyunsaturates are built into the cell membranes, resulting in reduced structural integrity or 'limpness', cholesterol is sequestered from the blood into the cell membranes to give them 'stiffness'. The problem was that there was no proof that lowering serum cholesterol levels could stave off CHD.

That did not prevent the American Heart Association calling for "modified and ordinary foods" useful for the purpose of facilitating dietary changes to newfangled oils away from traditional fats. These foods, said the AHA literature, should be made available to the consumer, "...reasonably priced and easily identified by appropriate labeling. Any existing legal and regulatory barriers to the marketing of such foods should be removed."

The man who made it possible to remove any "existing legal and regulatory barriers" was Peter Barton Hutt, a food lawyer for the prestigious Washington, DC, law firm of Covington and Burling. Hutt once stated: "Food law is the most wonderful field of law that you can possibly enter." After representing the edible oil industry, he temporarily left his law firm to become general counsel for the US Food and Drug Administration (FDA) in 1971.

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The regulatory barrier to foods useful to the purpose of changing American consumption patterns was the Food, Drug and Cosmetic Act of 1938, which stated: "...there are certain traditional foods that everyone knows, such as bread, milk and cheese, and that when consumers buy these foods, they should get the foods that they are expecting... [and] if a food resembles a standardized food but does not comply with the standard, that food must be labeled as an 'imitation'."

The 1938 Food, Drug and Cosmetic Act was signed into law partly in response to consumer concerns about the adulteration of ordinary foodstuffs. Chief among the products with a tradition of suffering competition from imitation products were fats and oils.

In his book, *Life on the Mississippi*, Mark Twain reports on a conversation overheard between a New Orleans cotton-seed oil purveyor and a Cincinnati margarine drummer. New Orleans boasts of selling deodorised cotton-seed oil as olive oil in bottles with European labels. "We turn out the whole thing—clean from the word go—in our factory in New Orleans... We are doing a ripping trade, too." The man from Cincinnati reports that his factories are turning out oleomargarine by the thousands of tons, an imitation that "you can't tell from butter". He gloats at the thought of market domination. "You are going to see the day, pretty soon, when you won't find an ounce of butter to bless yourself with, in any hotel in the Mississippi and Ohio valleys, outside of the biggest cities... And we can sell it so dirt cheap that the whole country has got to take it ... butter don't stand any show—there ain't any chance for competition. Butter's had its day—and from this out, butter goes to the wall. There's more money in oleomargarine than—why, you can't imagine the business we do."

In the tradition of Mark Twain's riverboat hucksters, Peter Barton Hutt guided the FDA through the legal and congressional hoops to the establishment in 1973 of the FDA 'imitation' policy

which attempted to provide for "advances in food technology" and give "manufacturers relief from the dilemma of either complying with an outdated standard or having to label their new products as 'imitation'... [since] ...such products are not necessarily inferior to the traditional foods for which they may be substituted". Hutt considered the word 'imitation' to be oversimplified, inaccurate and "potentially misleading to consumers". The new regulations defined 'inferiority' as any reduction in content of an essential nutrient that is present at a level of two per cent or more of the US Recommended Daily Allowance (RDA). The new 'imitation' policy meant that imitation sour cream, made with vegetable oil and fillers like guar gum and carrageenan, need not be labelled 'imitation' as long as artificial vitamins were added to bring macronutrient levels up to the same amounts as those in real sour cream. Coffee creamers, imitation egg mixes, processed cheeses and imitation whipped cream no longer required the 'imitation' label, but could be sold as real and beneficial foods, low in cholesterol and rich in polyunsaturates.

These new regulations were adopted without the consent of Congress, continuing the trend instituted under Nixon in which the White House would use the FDA to promote certain social agendas through government food policies. They had the effect of increasing the lobbying clout of special-interest groups such as the edible oil industry, and short-circuiting public participation in the regulatory process. It allowed food processing innovations, regarded as 'technological improvements' by manufacturers, to enter the marketplace without the onus of economic fraud that might be engendered by greater consumer awareness and congressional supervision. They ushered in the era of ersatz foodstuffs, convenient counterfeit products—weary, stale, flat and immensely profitable.

Congress did not voice any objection to this usurpation of its powers, but entered the contest on the side of the lipid hypothesis. The Senate Select Committee on Nutrition and Human Needs, chaired by George McGovern during the years 1973 to 1977, actively promoted the use of vegetable oils.

"Dietary Goals for the United States", published by the committee, cited USDA data on fat consumption and stated categorically that "the overconsumption of fat, generally, and saturated fat in particular...have been related to six of the ten leading causes of death" in the United States. The report urged the American populace to reduce overall fat intake and to substitute polyunsaturates for saturated fat from animal sources—margarine and corn oil for butter, lard and tallow.

Opposing testimony included a moving letter (buried in the voluminous report) by Dr Fred Kummerow of the University of Illinois, urging a return to traditional whole foods and warning against the use of soft drinks. In the early 1970s, Kummerow had shown that *trans* fatty acids caused increased rates of heart disease in pigs. A private endowment allowed him to continue his research, but government-funded agencies such as the National Institutes of Health refused to give him further grants.

One study that was known to McGovern Committee members, but not mentioned in its final report, compared calves fed saturated fat from tallow and lard with calves fed unsaturated fat from soybean oil. The calves fed tallow and lard did indeed show high-

er plasma cholesterol levels than the soybean-oil-fed calves; fat-streaking was found in their aortas, and atherosclerosis was also enhanced. But the calves fed soybean oil showed a decline in calcium and magnesium levels in the blood, possibly due to inefficient absorption. They utilised vitamins and minerals inefficiently, showed poor growth and poor bone development, and had abnormal hearts. More cholesterol per unit of dry matter was found in the aorta, liver, muscle, fat and coronary arteries—a finding which led the investigators to the conclusion that the lower blood cholesterol levels in the soybean-oil-fed calves may be the result of cholesterol being transferred from the blood to other tissues. The calves in the soybean oil group collapsed when forced to move around and they were unaware of their surroundings for short periods. They also had rickets and diarrhoea.

The McGovern Committee report continued dietary trends already in progress: the increased use of vegetable oils, especial-

ly in the form of partially hydrogenated margarines and shortenings. In 1976, the FDA established the GRAS (Generally Recognized As Safe) status for hydrogenated soybean oil. A report prepared by the Life Sciences Research Office of the Federation of American Scientists for Experimental Biology (LSRO-FASEB) concluded: "There is no evidence in the available information on hydrogenated soybean oil that demonstrates or suggests reasonable ground to suspect a hazard to the public when it is used as a direct or indirect food ingredient at levels that

are now current or that might reasonably be expected in the future."

When Mary Enig, a graduate student at the University of Maryland, read the McGovern Committee report, she was puzzled. Enig was familiar with Kummerow's research and she knew that the consumption of animal fats in America was not on the increase. Quite the contrary, the use of animal fats had been declining steadily since the turn of the century.

A report in the *Journal of American Oil Chemists*—which the McGovern Committee did not use—showed that animal fat consumption had declined from 104 grams per person per day in 1909 to 97 grams per day in 1972, while vegetable fat intake had increased from a mere 21 grams to almost 60 grams.¹⁴ Total per-capita fat consumption had increased over the period, but this increase was mostly due to an increase in unsaturated fats from vegetable oils—with 50 per cent of the increase coming from liquid vegetable oils and about 41 per cent from margarines made from vegetable oils.

Enig noted a number of studies that directly contradicted the McGovern Committee's conclusions that "there is...a strong correlation between dietary fat intake and the incidence of breast cancer and colon cancer"—two of the most common cancers in America. Greece, for example, had less than one-fourth the rate of breast cancer compared to Israel, but the same dietary fat intake. Spain had only one-third the breast cancer mortality of France and Italy, but the total dietary fat intake was slightly greater. Puerto Rico, with a high animal fat intake, had a very

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low rate of breast and colon cancer. The Netherlands and Finland both used approximately 100 grams of animal fat per capita per day, but breast and colon cancer rates were almost twice in the Netherlands what they were in Finland. The Netherlands consumed 53 grams of vegetable fat per person compared to 13 grams in Finland. A study from Cali, Colombia, found a four-fold excess risk for colon cancer in the higher economic classes which used less animal fat than the lower economic classes. A study found that Seventh Day Adventist physicians, who avoid meat (especially red meat), had a significantly higher rate of colon cancer than non-Seventh Day Adventist physicians.

Enig analysed the USDA data that the McGovern Committee had used and concluded that they showed a strong positive correlation with total fat and vegetable fat, and an essentially strong negative correlation or no correlation with animal fat to total cancer deaths, breast and colon cancer mortality and breast and colon cancer incidence. In other words, use of vegetable oils seemed to predispose to cancer, and

animal fats seemed to protect against cancer. She noted that the analysts for the committee had manipulated the data in inappropriate ways in order to obtain mendacious results.

Enig submitted her findings to the journal of the Federation of American Societies for Experimental Biology (FASEB) in May 1978, and her article was published in FASEB's *Federation Proceedings*¹⁵ in July of the same year—an unusually quick turnaround. The assistant editor responsible for accepting the article, died of a heart attack shortly thereafter. Enig's paper noted that the correlations pointed a finger at *trans* fatty acids and called for further investigation. Only two years earlier, the Life Sciences Research Office, which is the arm of FASEB that does scientific investigations, had published the whitewash that ushered partially hydrogenated soybean oil onto the GRAS list and removed any lingering constraints against the number-one ingredient in factory-produced food.

Enig's paper sent alarm bells through the industry. In early 1979 she received a visit from S. F. Reipma of the National Association of Margarine Manufacturers. Short, bald and pompous, Reipma was visi-

bly annoyed. He explained that both his association and the Institute for Shortening and Edible Oils (ISEO) kept careful watch to prevent articles like Enig's from appearing in the literature. Enig's paper should never have been published, he said. He thought that ISEO was "watching out". "We left the barn door open," he said, "and the horse got out."

Reipma also challenged Enig's use of the USDA data, claiming that it was in error. He knew it was in error, he said, "because we give it to them".

A few weeks later, Reipma paid a second visit, this time in the company of Tom Applewhite, an adviser to the ISEO and representative of Kraft Foods, Ronald Simpson with Central Soya, and a representative from Lever Brothers. They carried with them—in fact, waved in the air in indignation—a two-inch stack of newspaper articles, including one that appeared in the *National Enquirer*, reporting on Enig's *Federation Proceedings* article. Applewhite's face flushed red with anger when Enig repeated Reipma's statement that they had "left the barn door open and the horse got out" and his admission that

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Department of Agriculture food data had been sabotaged by the margarine lobby.

The other thing Reipma told Enig during his unguarded visit was that he had called in on the FASEB offices in an attempt to coerce them into publishing letters to refute her paper, without allowing Enig to submit any counter-refutation as was normally customary in scientific journals. He told Enig that he was "thrown out of the office"—an admission later confirmed by one of the FASEB editors.

Nevertheless, a series of letters did follow the July 1978 article.¹⁶ On behalf of the ISEO, Applewhite and Walter Meyer of Procter & Gamble criticised Enig's use of the data. Applewhite accused Enig of extrapolating from two data points, when in fact she had used seven.

John Bailar, Editor-in-Chief of the *Journal of the National Cancer Institute*, pointed out that the correlations between vegetable oil consumption and cancer were not the same as evidence of causation, and warned against changing current dietary components in the hope of preventing cancer in the future—which is, of course, exactly what the McGovern Committee did.

In reply, Enig and her colleagues noted that although the National Cancer Institute (NCI) had provided them with faulty cancer data, this had no bearing on the statistics relating to *trans* consumption and did not affect the gist of their argument—that the correlation with vegetable fat consumption, especially *trans* fat consumption, was sufficient to warrant a more thorough investigation. The problem was that very little investigation was being done.

University of Maryland researchers recognised the need for more research in two areas. One concerned the effects of *trans* fats on cellular processes once they are built into the cell membrane. Studies with rats, including one conducted by Fred Mattson in 1960, indicated that the *trans* fatty acids were built into the cell membrane in proportion to their presence in the diet, and that the turnover of *trans* in the cells was similar to that of other fatty acids. These studies, according to J. Edward Hunter of the ISEO, were proof that "*trans* fatty acids do not pose any hazard to man in a normal diet".

Enig and her associates were not so sure. Kummerow's research indicated that the *trans* fats contributed to heart disease; and

Kritchevsky, whose early experiments with vegetarian rabbits were now seen to be totally irrelevant to the human model, had found that *trans* fatty acids raise cholesterol in humans.¹⁷

Enig's own research, published in her 1984 doctoral dissertation, indicated that *trans* fats interfered with enzyme systems that neutralised carcinogens and increased enzymes that potentiated carcinogens.¹⁸

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Endnotes

1. Kritchevsky, D. et al., "Effect of Cholesterol Vehicle in Experimental Atherosclerosis", *Am. J. Physiol.* 178:30-32, July-September 1954
2. "Notice of Supelco-AOC Award to Kritchevsky", *Inform* 7:315, 1996
3. Enig, M. G., *Trans Fatty Acids in the Food Supply: A Comprehensive Report Covering 60 Years of Research*, Enig Associates, Inc., Silver Spring, MD, USA, 1995 (2ed), pp. 4-8
4. Groom, D., "Population Studies of Atherosclerosis", *Annals of Int. Med.* 55(1):51-62, July 1961; Enos, W. F. et al., "Pathogenesis of Coronary Disease in American Soldiers Killed in Korea", *JAMA* 158:912, 1955.
5. Laurie, W. et al., "Atherosclerosis and its Cerebral Complications in the South African Bantu", *Lancet*, February 1958, pp. 231-232

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6. Robertson, W. B., "Atherosclerosis and Ischaemic Heart Disease," *Lancet* 1:444, 1959
7. Gordon, T., "Mortality Experience Among Japanese in the US, Hawaii and Japan", *Pul. Health Rep.* 51:270, 1957; Pollak, O. J., "Diet and Atherosclerosis", *Lancet* 1:444, 1959
8. McGill, H. C. et al., "General Findings of the International Atherosclerosis Project", *Laboratory Investigations* 18(5):498, 1968
9. Smith, R. L. and E. R. Pinckney, *The Cholesterol Conspiracy*, Warren H. Green, Inc., St Louis, MO, USA, 1991, p. 125
10. De Bakey, M. et al., "Serum Cholesterol Values in Patients Treated Surgically for Atherosclerosis", *JAMA* 189(9):655-59, 1964
11. Keys, A., "Diet and Development of Coronary Heart Disease", *J. Chron. Dis.* 4(4):364-380, October 1956
12. Cristakis, G., "Effect of the Anti-Coronary Club Program on Coronary Heart Disease Risk-Factor Status", *JAMA* 198(6):129-35, November 7, 1996
13. "Dietary Goals for the United States—Supplemental Views", prepared by the Staff of the Select Committee on Nutrition and Human Needs, United States Senate, Government Printing Office, Washington, DC, November 1977, pp. 139-140
14. Rizek, R. L. et al., "Fat in Today's Food Supply—Level of Use and Sources", *J. Am. Oil Chem. Soc.* 51:244, 1974
15. Enig, M. G. et al., "Dietary Fat and Cancer Trends—A Critique", *Federation Proceedings* 37(9):2215-2220, FASEB, July 1978

16. Applewhite, T. H., "Statistical 'Correlations' Relating *Trans* Fats to Cancer: A Commentary", *Federation Proceedings* 38(11):2435-2439, October 1979
17. Kummerow, F. A., "Effects of Isomeric Fats on Animal Tissue, Lipid Classes and Atherosclerosis", *Geometrical and Positional Fatty Acid Isomers* (E. A. Emken and H. J. Dutton, eds), American Oil Chemists Society, Champaign, IL, USA, 1979, pp. 151-180; Kritchevsky, D., "Trans Fatty Acid Effects in Experimental Atherosclerosis", *Federation Proceedings* 41:2813, 1982
18. Enig, M. G., "Modification of Membrane Lipid Composition and Mixed-Function Oxidases in Mouse Liver Microsomes by Dietary *Trans* Fatty Acids", Doctoral Dissertation for the University of Maryland, 1984

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